2+ or greater MR. A recent German study[2] of 70 patients at 4 years reported an overall freedom from recurrence of mitral regurgitation grade 3+ or greater of 92% and a freedom from 2+ or greater MR of 71%, corroborating the earlier report. Our current findings align with these results, although our echocardiographic follow-up was not complete and 31% of patients had only 2+ MR preoperatively. Our patients had severely reduced LV function and predominantly central MR, which may be more reflective of greater symmetric LV and annular dilation. Patients with end-stage ventricles have been found to have more symmetric leaflet tethering and less jet asymetry.[3] It is difficult to compare the outcomes of patients with ischemic MR treated with ring annuloplasty, because ischemic MR may be associated with relatively preserved LV function, localized posterolateral infarct, and asymmetric posterior leaflet tethering or with end-stage ischemic cardiomyopathy with significant annular and LV dilation, symmetric leaflet tethering, and a centrally oriented regurgitant jet.[4] These extremes lie on a clinical continuum, both are associated with a ''normal'' appearing valve, and each is surgically corrected with undersized ring annuloplasty. We wholeheartedly agree with Hernández-Vaquero and colleagues that patient selection is paramount in optimizing surgical results, and as such we have chosen to restrict our use of the GeoForm prosthesis to patients with severe LV dysfunction, significant annular and chamber dilatation, and a central regurgitant jet. We believe that this form of ischemic MR is best treated with significant septolateral annular reduction, which is incorporated in the design of the GeoForm prosthesis. Although preoperative echocardiographic determination of leaflet tethering has been a focus of significant clinical research, leaflet and chordal tissue remodeling can also compensate for altered subvalvular alterations,[5] and mechanical forces insufficient to affect valvular competency induce geometric leaflet remodeling and altered gene expression.[6] Indeed, the unique adaptation of valvular tissue to ventricular remodeling is often forgotten when viewing echocardiographic studies of patients with end-stage dilated hearts and altered subvalvular geometry but little or no mitral regurgitation. The insightful analysis of the Asturias group has generated more questions than the limited answers our study can provide to the challenging problem of ischemic mitral regurgitation.

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EFFECT OF RIGHT VENTRICULAR FREE WALL VENTRICULOTOMY ON RIGHT VENTRICULAR FUNCTION: IS THAT THE CORRECT QUESTION?

To the Editor:

Lee and colleagues[1] recently reviewed the effects of limited versus conventional right ventriculotomy on subsequent right ventricular (RV) dilation and dysfunction in patients with tetralogy of Fallot who develop pulmonary regurgitation (PR) after transannular repair. No long-term benefits were seen after limited right ventriculotomy. The logic behind this strategy stems from the premise that the free wall RV incision generates dysfunction, with a corresponding hypothesis that late RV function would benefit from restricting the RV incision to less than 1 cm or avoiding it with transatrial and pulmonary artery approaches. Determining the reasons for such failure to offset long-term dysfunction requires analyzing relationships between RV structure and function. I therefore ask whether to use limited versus conventional right ventriculotomy is the right question. Questions about the advantages of limited versus conventional right ventriculotomy arose when knowledge of the relationships between RV structure and function was absent. We now know that the septum occupies 40% of ventricular muscle mass, dominates RV function, and is protected by standardized myocardial protection techniques in adults.[2]

The helical heart model of Torrent-Guasp shows that the RV free wall is predominantly composed of transverse fibers (basal loop) that cause circumferential compression (Figure 1), whereas the septum contains only helical fibers (apical loop) that produce twisting and shortening. Approximately 80% of RV function derives from longitudinal strain or shortening[3] and is quantified by tricuspid annular plane systolic

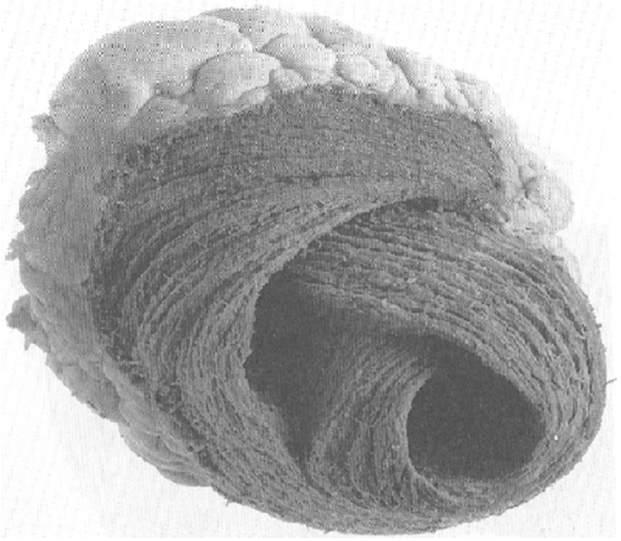

FIGURE 1. This illustration shows the fiber orientation relationships of the right ventricle whereby oblique fibers arising from the descending and ascending segments of the apical loop form the septum. The septum is surrounded by fibers with transverse muscular orientation arising from the basal loop, which comprises the free right ventricular wall.

excursion and its velocity. This movement follows septal helical coiling; "the septum is the RV lion."[4] Moreover, the limited contribution of the free wall is demonstrated by absence of RV failure when the entire free wall is cauterized, or replaced with a patch, provided that the septum is uninjured.[4] Conversely, septal damage causes RV failure, especially if pulmonary vascular resistance increases.

The long-term myocardial dilemma of PR is worsened by intraoperative septal muscle damage during tetralogy of Fallot repair. Chronic PR is not innocuous. Some septal dysfunction evolves from arterial fistulas after muscular ventriculectomy, but most damage stems from impaired myocardial protection. The incidence of intraoperative septal damage in children is unknown; however, 50% of 3300 adult patients were seen to develop septal paradox.[5] This complication is completely prevented by integrated myocardial protection.[2] The septum is the issue, *not* the free wall. This finding is underscored by novel procedures that include septal restoration when chronic PR is corrected.[4]

Development of surveys of intraoperative septal function in children is needed, especially because PR accentuates RV dysfunction; volume loading makes the RV cavity and wall more spherical, impairing septal function because it develops a more horizontal fiber angulation. Preventing PR by inserting outflow tract conduits with a valve may prevent pulmonary insufficiency, but focusing upon septal protection during the tetralogy of Fallot repair is important. Current myocardial protection patterns should be followed if postoperative septum function is normal but must change if septal injury develops, mirroring the adult cardiac surgical sequence.

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Reply to the Editor:

I appreciate the interest in our recently published study by Buckberg and Hoffman in their letter to the Editor. In our study, no long-term benefits were demonstrated for limited right ventricular (RV) incision relative to conventional (longer) RV incision in transannular repair of tetralogy of Fallot (TOF) in terms of RV volume and function. Buckberg and Hoffman speculate that our finding was due to the limited contribution of the RV free wall to the overall RV function. They stressed the importance of interventricular septum in maintaining normal RV function according to the helical heart model of Torrent-Guasp. I admit the limited contribution of the RV infundibulum, in which a transannular incision is made, to global RV function. As discussed in previous paper, Geva and colleagues[1] have demonstrated that the